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Brain Abscess: An Atypical CT Appearance of CNS Tuberculosis

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Brain abscess is an extremely rare manifestation of tuberculous involvement of the CNS; menigitis and tuberculoma are much more common [1-6]. The CT appearance of tuberculous abscess cannot be distinguished from many other entities that can present as a ring-enhancing lesion, such as pyogenic abscess, primary or metastatic neoplasm, or resolving hematomat. However, given the appropriate history one can suggest the correct diagnosis. We present an unusual case in which there was coexistence of supratentorial tuberculomas with an infratentorial tuberculous abscess.

Case Report

A 29-year-old man with productive cough, malaise, dysphagia, and weight loss was treated for pulmonary tuberculosis. Approximately 6 weeks after completing only 1 month of his projected 1-year course of antituberculous chemotherapy, he presented with continued fever, multiple cranial nerve dysfunctions, and prominent cerebellar findings. The patient was otherwise healthy, with no evidence of malnutrition or immunodeficiency. A chest radiograph demonstrated a large, thin-walled right apical cavity, fine nodular infiltrate in the left upper lobe, and no definite adenopathy.

Cranial CT before and after IV contrast infusion revealed a large, paravermian mass of low attenuation with a thin enhancing rim, compressing the fourth ventricle (Fig. 1). This was thought to represent a cerebellar abscess. Mild secondary hydrocephalus was also present. Additionally, there were several homogeneously enhancing supratentorial masses (Fig. 2) consistent with tuberculomas. There was no contrast enhancement of the basilar cisterns.

The large cerebellar abscess with a firm capsule was surgically drained through an occipital craniotomy. The purulent contents were positive for acid-fast bacilli. Cultures from the abscess revealed Mycobacterium tuberculosis. The patient recovered with antituberculous medication.

Discussion

CNS tuberculosis presents most frequently as basilar meningitis, followed by tuberculomas [1, 5, 6]. True tuberculous brain abscess is extremely rare [2-4] and almost all cases were reported in the pre-CT era. CNS tuberculosis is thought to arise from hematogenous spread of the tubercle bacillus from a primary site of infection [3, 6]; however, up to 30% of cases have no prior or current evidence of extracranial infection [1].

In Whitener's exhaustive review of the literature [3], there were only 17 verified cases of tuberculous brain abscess. His strict criteria included macroscopic evidence of capsule formation with a pus-containing cavity, inflammatory reaction in an abscess wall composed primarily of vascular granulation tissue, and proof of tuberculous origin by either culture or acid-fast stain. The central multinucleate giant cells surrounded by epithelioid granulomatous reactions that characterize tuberculomas are absent in tuberculous abscess [3, 4].

The pathogenesis of abscess formation is thought to be related to multiple factors, including host sensitivity, size of inoculation, and protein and vitamin deficiencies [2-4]. It has been suggested that the influx of polymorphonuclear leukocytes into an area of massive caseous necrosis in a hypersensitive individual might lead to liquefaction and abscess formation [7].

Differentiation between tuberculomas and tuberculous abscess can be difficult when using CT criteria alone. A tuberculoma can appear as a nodular or ring-enhancing mass [8-12]. Calcification occurs in only 1-6% [2, 11, 12]. Tuberculomas may be diagnosed with a high degree of confidence when there is a “target sign”—a central area of calcification with peripheral ring-enhancement [9]. It has also been stated that the central area of caseous necrosis in a tuberculoma is commonly isodense with brain tissue, whereas the central area of pus in an abscess is decreased in attenuation [9]. Besides histologic and CT differences, tuberculous abscess has a more rapid clinical course, is usually larger in size, and is said to occur in older age groups as compared with tuberculomas [2, 3, 12].

Because of the history of pulmonary tuberculosis in our patient, the homogeneously enhancing, relatively small supratentorial lesions were thought to represent tuberculomas. The ring-enhancing posterior fossa lesion had the CT characteristics of a brain abscess. Even though bacterial superinfection of tuberculomas has been reported in the preantibiotic era [8], it is important to raise the possibility of tuberculous
abscess since appropriate cultures and stains must be obtained at the time of surgery.

The presentation of CNS tuberculosis in our patient was undoubtedly related to the incomplete course of chemotherapy. However, he had no known predisposing factors making him more susceptible to abscess formation. Because tuberculomas and a tuberculous abscess were both present, one might postulate that the abscess arose from a larger posterior fossa tuberculoma with an exaggerated exudative reaction. Alternatively, differences in intraparenchymal presentation might have been related to the size of each inoculation.

In the past, mortality in patients with tuberculous brain abscess has been reported to be approximately 40% [3, 4]. Earlier diagnosis and treatment with the aid of CT scanning should reduce this figure. The successful treatment in all reported cases involved both surgical intervention and adequate medical therapy [3].

In summary, tuberculous brain abscess is rare and the CT appearance cannot be distinguished from other entities that can cause a ring-enhancing lesion. However, this diagnosis can be suggested in the appropriate clinical setting, particularly when other CT evidence of tuberculosis is present.

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REFERENCES